

# Emergency Treatment of Haemorrhaging Coeliac or Mesenteric Artery Aneurysms and Pseudoaneurysms in the Era of Endovascular Management

K.J. Roberts <sup>a</sup>, N. McCulloch <sup>a</sup>, C. Forde <sup>b</sup>, B. Mahon <sup>b</sup>, K. Mangat <sup>b</sup>, S.P. Olliff <sup>b</sup>, R.G. Jones <sup>b,\*</sup>

<sup>a</sup> Department of Hepatobiliary and Pancreatic Surgery, Queen Elizabeth Hospital Birmingham, Birmingham, UK

<sup>b</sup> Department of Radiology, Queen Elizabeth Hospital Birmingham, Birmingham, UK

## WHAT THIS PAPER ADDS

This is the first paper to describe the treatment and outcomes of an unselected consecutive series of symptomatic visceral artery aneurysms where interventional radiology has been the primary modality. It documents a 5-year period at a high-volume specialist centre, with at least 12 months of follow-up. Morbidity and aneurysm-related mortality are compared with studies where up-front surgery was the treatment of choice.

**Objectives:** Patients requiring emergency treatment of visceral artery aneurysms (VAAs) can be treated by endovascular or surgical techniques. Outcomes after failed attempts at endovascular control are unclear as is the present role of surgery. This study reviewed treatment and outcomes of a contemporary cohort of patients with symptomatic VAAs at a tertiary referral centre.

**Methods:** Patients undergoing emergency treatment of a VAA of the coeliac, mesenteric arteries, or their branches were identified over a 5-year period. Patient variables, treatments, and outcomes were assessed.

**Results:** Forty-eight patients underwent 65 radiological and two surgical procedures. Pseudoaneurysms were present in 45 (94%) of patients. Interventional radiology procedures were the initial treatment in every patient. The initial success was 40 out of 48 (83%). Patients requiring more than one procedure were all successfully treated. Regarding initial failures, if the VAA sac could not be accessed at angiography an alternative procedure to control the VAA was required in every case. If initial endovascular treatment failed, repeating the same procedure was successful in half of the patients. Ultrasound-guided percutaneous VAA embolisation was used in four patients. The 30-day mortality was eight out of 48 (17%). There were four recorded complications including one death directly attributable to VAA treatment.

**Conclusions:** Patients needing emergency treatment of a VAA could be well served by non-surgical management. When the initial attempt at control of bleeding is unsuccessful it is important to consider non-conventional means of accessing these arteries. The need for surgery, in selected centres, may exist for a small group of patients after initial failed radiological treatment only.

© 2015 European Society for Vascular Surgery. Published by Elsevier Ltd. All rights reserved.

Article history: Received 19 March 2014, Accepted 13 December 2014, Available online 23 February 2015

**Keywords:** Pseudoaneurysm, Endovascular treatment, Visceral artery aneurysm

## INTRODUCTION

Visceral artery aneurysms (VAAs) comprising true and pseudoaneurysms are rare,<sup>1</sup> with a reported incidence of 0.1–2%,<sup>2</sup> and have the potential to cause life-threatening haemorrhage if ruptured.<sup>3,4</sup> Aetiology is varied and often related to the vessel involved. Historical treatment of VAA involved surgical management<sup>3</sup> but has evolved towards

less invasive techniques due to decreased morbidity and mortality.<sup>4</sup> Conservative management, which may include observation of asymptomatic lesions, is now widely used. Symptomatic VAAs however typically present with signs of haemorrhage, cannot be simply observed, and require emergency treatment. The treatment and outcomes of patients undergoing emergency treatment is different from that for patients with asymptomatic VAA and thus cannot be directly compared. Almost all previous studies have grouped these patients together,<sup>2,5–15</sup> however, making interpretation of treatment and outcomes unclear.

Angiography with a view to embolisation or stent-graft insertion is the main endovascular treatment option to exclude these symptomatic aneurysms. These treatments benefit from low procedural morbidity and mortality and have been shown to be highly successful but require specialist personnel and equipment.<sup>7,15</sup> Recent studies present a wide

<sup>\*</sup> Presented at the European-African Hepato-Pancreato-Biliary Association, Belgrade, May 2013 and at the Association of Surgeons of Great Britain and Ireland, Glasgow, May 2013.

\* Corresponding author. Robert G Jones, Queen Elizabeth Hospital Birmingham, University Hospitals Birmingham NHS Trust, Mindelsohn Way, Edgbaston B15 2TH, UK.

E-mail address: [robert.jones@uhb.nhs.uk](mailto:robert.jones@uhb.nhs.uk) (R.G. Jones).

1078-5884/© 2015 European Society for Vascular Surgery. Published by Elsevier Ltd. All rights reserved.

<http://dx.doi.org/10.1016/j.ejvs.2014.12.019>

variety in the proportion of patients treated with endovascular approaches or surgery as their initial management. At one extreme, surgery was used in a few or no patients<sup>5–7</sup> and at the other in the majority.<sup>2,8,13</sup> If initial endovascular management is unsuccessful patients can be managed with repeated efforts at endovascular control, open surgery, or rarely alternative minimally invasive techniques. There are case reports of transabdominal ultrasound or endoscopic ultrasound (EUS)-guided VAA injection and embolisation after failed endovascular treatment,<sup>7,16,17</sup> although outcomes among large consecutive series are lacking. Differing practice between units, the period over which the study was conducted, and the varying numbers of patients with asymptomatic/symptomatic VAA in these studies renders assessment of the current emergency treatment of symptomatic VAA unclear.<sup>2,5,6,8–10,12,13,15,18</sup> Endovascular management is well suited to the treatment of symptomatic lesions given the minimally invasive nature of the treatment and avoidance of surgery in inflamed tissues with major haematoma infiltration.

There are also few data to show the outcomes after initial failed attempts at endovascular treatment.<sup>9,11</sup>

The aim of this study therefore was to review the experience and outcomes of a contemporary cohort of patients undergoing emergency treatment of a VAA at a high-volume tertiary referral centre with a 24-hour interventional radiology service. Treatment and outcomes were identified, in particular the need for more than one procedure, for non-endovascular treatment, and complications.

## METHODS

This was a retrospective review of all patients with a VAA of the coeliac or mesenteric arteries at a single high-volume centre. Workload within this centre includes tertiary referral services to provide liver transplantation, hepatobiliary and pancreatic surgery, sarcoma surgery, vascular surgery, and general surgery. All patients undergoing emergency treatment (endovascular or surgical) of a symptomatic VAA were included. Symptomatic VAAs were defined as those presenting with gastrointestinal haemorrhage (haematemesis, malaena, or haemobilia) and peritoneal or retroperitoneal haemorrhage due to the presence of the VAA. Owing to the different treatment strategies and outcomes, patients with asymptomatic VAA were excluded from the study.

Patients with VAAs were identified by searching all electronic hospital radiology reports (both cross-sectional imaging and interventional radiological procedures) for the keywords “aneurysm”, “pseudoaneurysm”, or “embolisation”. All positive reports were manually checked to verify the nature of pathology and whether the patient was being treated for a symptomatic VAA. It is possible that this strategy might miss some patients whose computed tomography (CT) was performed at the local hospital and then underwent surgical treatment. However many “outside” CT reports are re-reported at this institution and patients requiring surgery would have cross-sectional imaging at some point to assess adequacy of treatment. Radiology

reports include an electronic description of the patient’s history and previous treatment. Thus the only group of patients at risk of not being identified are those with an unreported outside CT scan undergoing surgical treatment and dying without any postoperative radiological study within the institution. Patient hospital records and electronic data (clinical observations, blood test results, operation notes, radiology procedures) were then reviewed individually. Patients with a VAA of the coeliac, superior mesenteric, or inferior mesenteric arteries or their branches were included. Patients with renal or other artery VAAs were not included. Subsequent treatment (surgery or interventional radiology) was reviewed together with outcomes, procedural failure/success, the need for further intervention, complications, and mortality. The study period was 5 years from January 1, 2008, to December 31, 2012. There was a minimum 12-month follow-up for each patient. Follow-up consisted of clinical review at 1, 3, 6, and 12 months (dependent upon the post-treatment clinical course). A follow-up CT scan was typically performed at 3 months following all treatment to review the VAA.

The anatomical site, aetiology, treatment at the referring institution, number and type of procedures, complications and outcome were recorded. All radiology procedures were performed by dedicated consultant interventional radiologists.

## Treatment algorithms

Patients were assessed and resuscitated using principals of permissive hypotension.<sup>19</sup> If a patient was haemodynamically stable and with no evidence of contrast extravasation at CT angiography they would typically undergo treatment during the daytime; however, active extravasation of contrast or haemodynamic instability would be treated at that time. Once haemodynamically stable, if possible, with or without general anaesthesia, patients would undergo a CT scan with arterial enhancement in order to define and understand the anatomy of the VAA prior to percutaneous embolisation. If a bleeding site was identified, patients were then moved to the endovascular suite for intervention. Occasionally, patients too unwell for CT scan would proceed directly to percutaneous angiography. The transfemoral approach was used in all patients undergoing angiography. This route permits a wide variety of catheters of different shapes/sizes to selectively access the mesenteric vessels. Angiography was then carried out with injection of iodinated contrast through the catheter into the vessel to delineate the vessel and any associated pathology. From here embolisation materials (e.g. coils/glue) can be injected through the catheter under fluoroscopic guidance to treat the aneurysm or pseudoaneurysm. Prior to embolisation safe catheter tip position is essential to prevent non-target embolisation of adjacent normal structures.

Coils are the first choice of material to achieve embolisation with a typical 10% oversize in coil size compared with the diameter of the vessel. Coil length depends on the anatomy but frequently used lengths were 7 cm or 14 cm (Cook medical Nestor coils, Bloomington, IN, USA). Smaller length

coils were also available however. Histoacryl glues were used selectively particularly when the body of the aneurysm or pseudoaneurysm could be selectively catheterised rather than injecting in the parent artery especially in the gastrointestinal tract vasculature. Polyvinyl alcohol particles are rarely used in treating aneurysms or pseudoaneurysms.

In some patients, aneurysms (typically those with lesions that could not be adequately treated by angiographic techniques) were treated by direct percutaneous or endoscopic injection. There was no departmental protocol for this, and so these cases are described in more detail in the results section. Following the procedure, patients were cared for in a high-dependency or intensive care setting. Follow-up CT scans were performed to ensure satisfactory treatment of the VAA in every case. Recurrent episodes of hypotension, gastrointestinal bleeding, or falls in haemoglobin triggered emergent CT imaging or repeated angiography.

Data describing the cohorts were expressed as the median (interquartile range) or absolute number and percentage. The primary outcome of interest was 30-day mortality defined as the time from first intervention at our institution.

### Definitions

In this manuscript interventional radiology procedures refer to all angiographic and percutaneous procedures performed under ultrasound or CT guidance.

## RESULTS

### Cohort characteristics

Forty-eight patients were identified, with 40 referred from outside of our centre; three had treatment at the referring hospital prior to transfer (endoscopic injection to a bleeding duodenal ulcer, transarterial coil embolisation  $\times$  2). Patient variables, method of presentation, aetiology, and anatomical site of the VAA are summarised in Table 1.

### Initial treatment and the need for further procedures

Interventional radiology was the initial treatment in all patients: endovascular procedures  $n = 47$  and ultrasound guided percutaneous injection  $n = 1$  (of an intrahepatic VAA). Active bleeding was observed in 16 patients (33%).

All endovascular procedures were performed via the common femoral artery. The radiological interventions were as follows (some patients received more than 1 method of embolisation): coils  $n = 38$ , glue  $n = 9$ , thrombin injection  $n = 4$ , polyvinyl alcohol particles  $n = 3$ , gel foam  $n = 1$ . Stent-grafts were used in two patients (both 8-mm Viabahn stents). Four aneurysms could not be successfully treated at this initial attempt due to the inability to obtain a catheter position to treat the VAA in three patients, and no possible endovascular treatment was possible in one patient (without occlusion of common hepatic artery post liver transplant). All these patients required further treatment.

A further four patients required a second procedure (total  $n = 8$ , 17%; Table 2). The indication for the second

**Table 1.** Summary of cohort characteristics, presentation, aetiology and anatomical location of the VAA.

Characteristic	Whole cohort $n = 48$	Need for repeat procedure $n = 8$	30 day mortality $n = 8$
Male gender $n$ (%)	30 (63%)	5	5
Age median (IQR)	61 (43–70)		
Pseudoaneurysm	45 (94%)	8	7
<b>Presentation</b>			
Haemorrhage	48 (100%)	8	8
<b>Aetiology</b>			
Trauma	13	2 ( $s = 1$ )	0
Pancreatitis	12	0	3
Idiopathic	6	2	2
Biliary sepsis	5	1	1
Post biopsy/TIPPS	3	0	1
POPF	3	0	1
Post liver transplant	2	1 ( $s = 1$ )	0
Peptic ulcer	2	0	0
Mycotic	1	1	0
Coeliac artery stenosis	1	1	0
<b>Site of VAA</b>			
Common hepatic	1	1 ( $s = 1$ )	0
True hepatic	1	0	1
Right hepatic	14	1	2
Left hepatic	2	2	0
Intrahepatic	8	1	0
Dorsal pancreatic	1	0	1
Splenic	7	0	1
Intrasplenic	2	1	0
Gastroduodenal	7	0	1
IPDA	2	1	1
Jejunal/ileal	3	1 ( $s = 1$ )	1

Note. ( $s = n$ ) denotes the numbers of patients undergoing surgical treatment. IQR = interquartile range; TIPPS = transjugular intrahepatic portosystemic shunt; POPF = postoperative pancreatic fistula; IPDA = inferior pancreaticoduodenal artery; VAA = visceral artery aneurysm.

procedure among these four patients was further haemorrhage. Six of these eight patients were treated with further interventional radiology procedures (4 of whom had ultrasound-guided embolisation). Of the three patients in whom the VAA was not accessible at initial angiography, all required a non-endovascular technique to control the lesion. In those with an initial failed endovascular procedure or missed bleeding point ( $n = 3$ ) subsequent repeat endovascular treatment was successful in two patients.

Surgical intervention was required in two patients (4%). In the first patient bleeding occurred from a VAA of a caecal branch of the ileocolic artery 2 weeks following severe blunt polytrauma. At angiography no active bleeding was seen, and coiling of what was thought to be the responsible VAA was performed. Subsequently further gastrointestinal haemorrhage was treated with a right hemicolectomy. In the second patient, open surgical excision and repair of a common hepatic artery VAA was required 1 month post liver transplant. Two further patients subsequently developed a new VAA at a different site.

**Table 2.** Summary of patients requiring more than one procedure.

Patient summary	Aetiology	Artery	Initial technique	Reason for repeat procedure	Further treatment	Outcome
74 M	Inflammatory/idiopathic	Left hepatic	Endovascular	Unable to gain access: tortuous or aberrant anatomy	EUS thrombin injection	Well, no further bleed
73 F	Coeliac artery stenosis	IPDA	Endovascular	Unable to gain access: tortuous or aberrant anatomy	Endovascular $\times$ 1 (failure as still not able to gain access to PA); EUS histoacryl glue injection	Well, no further bleed
81 F	Idiopathic	Left hepatic	Endovascular	Unable to gain access: tortuous or aberrant anatomy	US thrombin injection $\times$ 2	Well, no further bleed
26 M	Trauma	Ileocolic	Endovascular	Failure of initial control: coil embolisation of PA	Surgery: right hemicolectomy	Well, no further bleed
76 M	Bile duct injury	Right hepatic	Endovascular	Failure of initial control: coil embolisation of PA	Endovascular coil and lipiodol injection	Alive; Liver abscess; no further bleed
66 M	Mycotic: infective endocarditis	Intra hepatic	US thrombin injection	Failure of initial control: thrombin injection — non-obiterated VAA seen at CECT $\times$ 2	US thrombin injection $\times$ 2	Well, no further bleed
71 M	Trauma	Intra splenic	Endovascular	Missed bleeding site: short gastrics embolised initially; subsequent bleed from intrasplenic artery	Endovascular coil embolisation	Well, no further bleed
42 F	Post liver transplant	Common hepatic	Endovascular	No endovascular options without complete occlusion of HA	Surgery - arterial reconstruction	Died 2.3 years later: developed HAT

VAA = visceral artery aneurysm; IPDA = inferior pancreaticoduodenal artery; EUS = endoscopic ultrasound; US = ultrasound, CECT = contrast enhanced computerised tomography; HAT = hepatic artery thrombosis.

### Patients managed by non-endovascular, non-surgical treatments

**Patient 1.** A stent-graft was placed across the origin of the segment iv hepatic artery (which gave subsequent rise to the intrahepatic VAA). Follow-up CT demonstrated persistent VAA filling and following a re-bleed the patient had a successful peripheral ultrasound-guided thrombin injection to the VAA.

**Patient 2.** A VAA of the inferior pancreaticoduodenal artery, in a patient with coeliac artery occlusion, required transduodenal endoscopic ultrasound injection of histoacryl glue after two failed attempts at endovascular control (Fig. 1Ba–c).

**Patient 3.** A VAA of the left hepatic artery was treated by two percutaneous injections of thrombin under ultrasound guidance after a failed attempt of endovascular control.

**Patient 4.** A VAA of the right hepatic artery required a second percutaneous thrombin injection with coils under ultrasound guidance after initial failed attempt at percutaneous injection.

### Outcomes and complications

The median follow-up was 283 days (IQR 44–804), and at last follow-up 13 (27%) patients had died. The 30-day mortality was eight out of 48 (17%, summarised in Table 3). Among these it was not possible to control haemorrhage in two patients who were too frail for other treatment, and small bowel infarction secondary to embolisation of glue occurred in one patient. The remainder died due the underlying disease process with no evidence of ongoing VAA haemorrhage at or before the time of death.

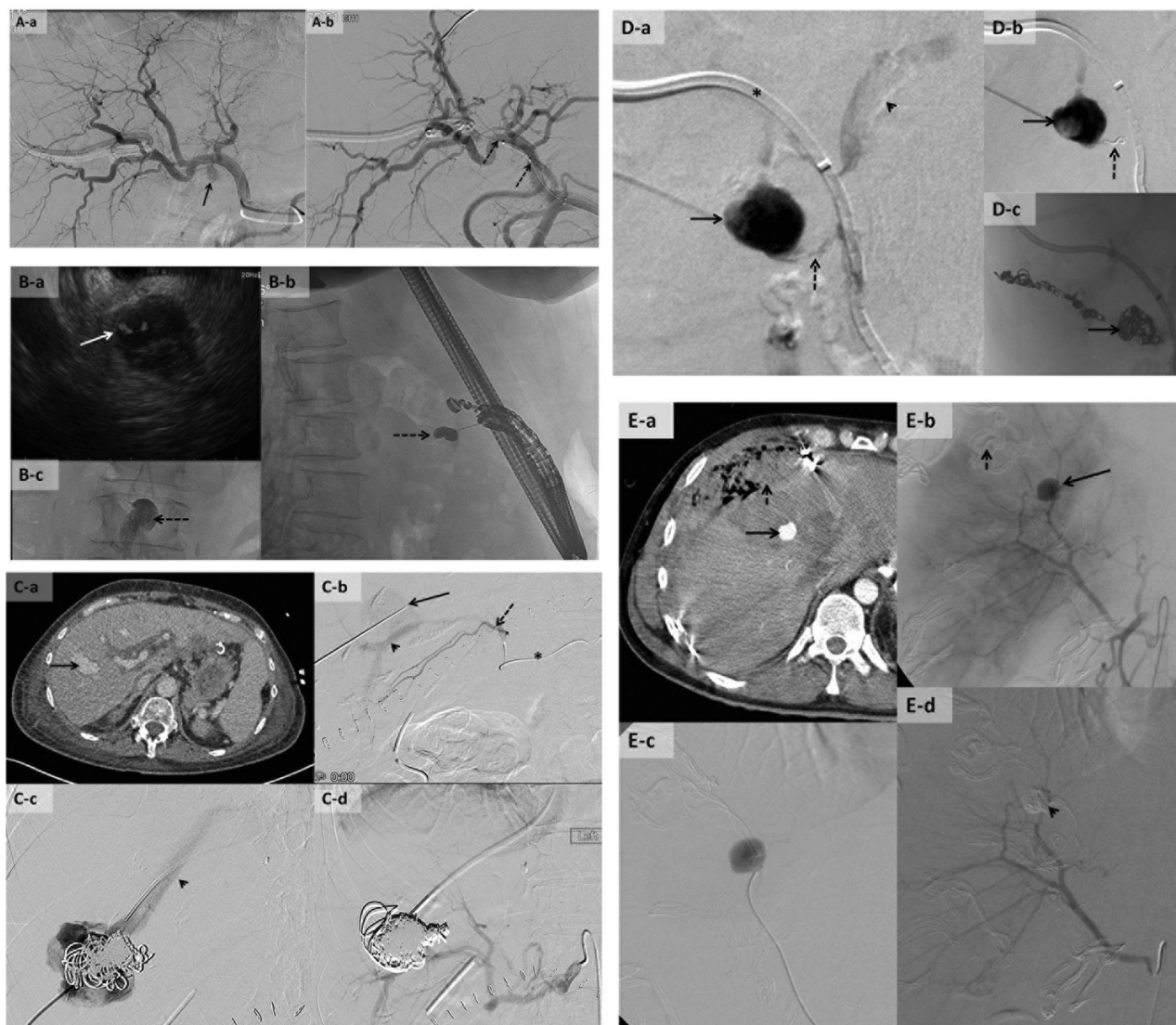
Regarding the five deaths occurring after 30 days, three were of unknown causes after discharge, one of unrelated causes after discharge, and one of late hepatic artery thrombosis 2 years after hepatic artery reconstruction for post liver transplant VAA of the hepatic artery.

Four complications, including one death, were related to interventional radiology procedures and occurred following 65 procedures (6% of interventions or 8% of patients). One patient with multi-organ failure secondary to necrotising pancreatitis developed small bowel infarction due to glue migration; one patient with a splenic artery VAA had complete splenic infarction but did not require splenectomy; one patient with a right hepatic artery VAA developed a liver abscess following right lobe infarction requiring antibiotic therapy; and one patient with an intrahepatic VAA developed a liver haematoma without clinical consequence.

### DISCUSSION

This study reports the outcomes of consecutive patients with symptomatic VAA undergoing emergency treatment at a single centre with a dedicated interventional radiology service as well as vascular, hepatobiliary/pancreatic/liver transplant, and general surgery teams. The main findings were that all initial attempts at treatment were non-surgical and surgical treatment was required in less than 5% of





**Figure 1.** Examples of visceral artery aneurysms (VAAs) requiring treatment other than routine endovascular coil embolisation. Patient A: endovascular stent. An idiopathic VAA of the right hepatic artery (A-a, solid arrow) could only be occluded by placement of an endovascular stent (A-b, broken arrows). Patient B: EUS-guided injection. An IHPDA VAA after failed attempt at endovascular approaches. The VAA is seen (B-a, bold arrow) at EUS and treated by direct injection of 3.5 ml histoacryl glue (B-b and -c, broken arrow). Patient C: peripheral coil embolisation. Haemorrhage two weeks following liver transplantation. The VAA is seen (C-a and -b, bold arrow). Arterial contrast via the endovascular catheter (C-b, \*) fills arterial branches (C-b, broken arrow), a portal vein branch (C-b, arrow head) and a hepatic vein (C-c, arrow head). Extensive percutaneous coil embolisation was required to completely occlude the VAA (C-c and -d). Patient D: peripheral coil embolisation and coil of fistula track. The VAA is seen (bold arrow) as is an arteriobiliary fistula (D-a, broken arrow) with contrast seen in the biliary tree and left hepatic duct (D-a, arrow head). An external biliary drain is present (\*). A microcoil successfully occluded the fistula (D-b, broken arrow). The VAA was then embolised with coils as was the needle track (D-c). Patient E: endovascular injection. The VAA is seen (E-a and -b, bold arrow) as are liver packs (E-a and -b, broken arrow) used to control haemorrhage following blunt trauma. The endovascular catheter went straight into the VAA (E-c) facilitating direct injection of 1.5 mL of histoacryl glue/lipiodol mixture. The glue cast is seen in E-d (arrow head).

patients. Procedural morbidity and mortality were present but represented a small proportion of the overall morbidity and mortality due to the underlying disease processes, and thus interventional radiology for VAAs appears to be a successful strategy with acceptable results. These results, however, need to be considered in the context of this specialist service at this high-volume tertiary referral centre.

No patient underwent surgery as an initial procedure reflecting the shift in treatment of these lesions (a summary

of recent studies reporting the treatment and outcome of patient cohorts with VAA is provided in the [Supplementary Table 1](#)). Reports of endovascular repair have typically excluded surgical patients<sup>6,7,9,12,20–22</sup> and those that include them have reported varying numbers of patients initially treated with surgery.<sup>2,13,15,23</sup> Despite widespread use of endovascular treatments there are units with recently published series where the majority of patients have been treated with surgery.<sup>2,8,10,13</sup> In the present

**Table 3.** Summary of patients who died during the hospital procedure associated with VAA treatment.

Patient summary	Aetiology	Artery	Summary of investigations/treatment	Cause of death
39 F	Alcoholic pancreatitis	Dorsal pancreatic	Embolisation of glue and thrombosis of SMA	Small bowel infarction secondary embolisation
70 F	Gall stone pancreatitis	IPDA	No options for endovascular control and too frail for surgery	Uncontrollable bleeding
75 M	Bile duct injury	Right hepatic	Further bleed after 3 weeks; patient in MOF and too frail for further treatment	Uncontrollable bleeding and MOF/frailty
18 M	Alcoholic pancreatitis	Gastro- duodenal	Severe necrotising pancreatitis and MOF	MOF related to pancreatitis
65 M	Liver biopsy	Right hepatic	Aggressive liver tumour with no evidence of bleeding	Malignant disease
84 M	Idiopathic	Common hepatic	Frail and elderly patient developed MOF including liver dysfunction.	MOF related to liver dysfunction and patient frailty
69 M	POPF	Splenic	Grade C POPF; developed VAA and MOF	MOF related to systemic sepsis
86 M	Idiopathic	Jejunal branch	Myocardial infarction on 7th post procedure day with no evidence of haemorrhage	Myocardial infarction

VAA = visceral artery aneurysm; IPDA = inferior pancreaticoduodenal artery; MOF = multi-organ failure; POPF = post operative pancreatic fistula; VAA = visceral artery aneurysm.

cohort of consecutive patients requiring emergency treatment of VAA, all were initially treated with interventional radiology procedures with surgery reserved for failures only. Those centres predominately performing up-front surgery (for a mix of elective and emergency VAAs) demonstrate overall low rates of morbidity and mortality. Mortality can reach 20–25% following surgery for bleeding VAAs<sup>8,18</sup> however. The case for upfront surgery will be dictated by the available interventional radiology service. Units with high rates of success with endovascular procedures are those with the lowest rates of surgical intervention (ref. 15 and the present study). In the largest published series of endovascular treatment no patient underwent surgical treatment after initial endovascular failure.<sup>7</sup> Unfortunately, it is not clear how many patients were treated with up-front surgery as they were excluded from that study.

The 30-day mortality in the present series is comparable with other series of predominately/sole endovascular repair. Case mix makes direct comparison difficult; however, the vast majority of reported deaths are in patients with haemorrhage. The present study includes only patients with symptomatic VAA and haemorrhage thus mortality and morbidity appear satisfactory. It is difficult to draw clear conclusions regarding the role of surgery and endovascular treatment in this subgroup of emergency patients given that this is a single-centre study where so few patients underwent surgical treatment. The management of these patients in this institution is to proceed directly to angiography (with a prior CT angiogram if the patient is haemodynamically stable) with or without intervention and then select following treatment (if needed) on a case-by-case basis with discussion between the relevant surgeons and interventional radiologists. Further studies including patients only undergoing emergency treatment are required to compare treatments and outcomes between units.

Nearly one in six patients did require more than one procedure, however. The reasons for this were varied, but the most common could be grouped into two distinct categories: failure to access the VAA radiologically or failure of initial attempts at haemorrhage control. In the first setting successful control could only be achieved by alternative means (ultrasound-guided injection and surgical repair) and in the second scenario repeated attempts using the same strategy seem justified as this yielded a satisfactory result in half of these cases. For those patients where initial attempts at endovascular control failed, the use of ultrasound-guided VAA injection was an alternative treatment with no morbidity and success in every case. One patient did require a second thrombin injection to complete occlusion of the VAA, 24 hours after initial injection. Some of these interventional procedures where treatment other than routine endovascular embolisation was required are demonstrated in Fig. 1. It is important to note that the above groups consist of small patient numbers and thus conclusions drawn must be interpreted in this light. However, with no prospective or randomised trials of treatment of VAAs upon which to draw conclusions, retrospective series such as this are essential to report outcomes of contemporary and modern management

approaches of uncommon diseases such as VAAs. Future studies could be improved by reporting multicentre outcomes and by the development of a prospective multicentre database.

Other groups have debated criteria upon which to select VAA for treatment<sup>7,15</sup>; the main point of discussion is whether historical criteria used to select patients for surgical repair<sup>14,24</sup> are applicable in the era of endovascular treatment. Clearly symptomatic VAAs do require treatment and it appears that endovascular repair is associated with low rates of morbidity, mortality, and high success. Those studies with a high proportion of asymptomatic patients reporting elective endovascular repair show lower morbidity and no mortality in that setting.<sup>7,15</sup> It has been reported that pseudoaneurysms have a higher risk of bleeding than true aneurysms,<sup>9,25</sup> that the size of pseudoaneurysm does not correlate with bleeding risk,<sup>26</sup> and that bleeding risk is related to the artery involved.<sup>9</sup>

With no robust trials involving treatment of symptomatic VAAs upon which to draw conclusions, retrospective series such as this are essential to report outcomes of contemporary and modern management approaches of uncommon diseases such as VAAs. Future studies could be improved by reporting multicentre outcomes and by the development of a prospective multicentre database.

Weaknesses of the study include confounding due to local referral patterns and its retrospective nature. However, this problem is likely to affect all published series to some degree as they present data from specialist centres. With the retrospective study design it is possible that some patients with VAAs were missed. Since the use of radiology is common to all hospital speciality teams and that cross-sectional imaging is essential to understand the anatomy of VAAs and the post-procedure/postoperative management of patients with VAAs, by searching all electronic radiology records for keywords all patients with a VAA should have been identified.

## CONCLUSION

At this tertiary referral centre with a dedicated interventional radiology service, all patients requiring emergency treatment of their VAA were initially treated with interventional radiology procedures. Success was high and procedural morbidity and mortality appear to be low in this setting though repeated procedures were not uncommon. Further studies are required to review the present role of interventional radiology and surgical procedures and their outcomes in services with different levels of access to interventional radiology services. It is essential that patients requiring elective or emergency treatment are separated to allow interpretation of these results.

## CONFLICT OF INTEREST

None.

## FUNDING

None.

## ACKNOWLEDGEMENTS

The authors would like to acknowledge the work of Ravi Marudanayagam, Robert P. Sutcliffe, Thamara Perera, Hynek Mergental, Simon Bramhall, Paolo Muiesan, John Isaac, and Darius Mirza in collecting the data that helped facilitate this study.

## APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.ejvs.2014.12.019>

## REFERENCES

- 1 Hossain A, Reis ED, Dave SP, Kerstein MD, Hollier LH. Visceral artery aneurysms: experience in a tertiary-care center. *Am Surg* 2001;**67**(5):432–7.
- 2 Pulli R, Dorigo W, Troisi N, Pratesi G, Innocenti AA, Pratesi C. Surgical treatment of visceral artery aneurysms: a 25-year experience. *J Vasc Surg* 2008;**48**(2):334–42.
- 3 Carr SC, Pearce WH, Vogelzang RL, McCarthy WJ, Nemcek Jr AA, Yao JS. Current management of visceral artery aneurysms. *Surgery* 1996;**120**(4):627–33.
- 4 Sachdev-Ost U. Visceral artery aneurysms: review of current management options. *Mt Sinai J Med* 2010;**77**(3):296–303.
- 5 Balderi A, Antonietti A, Ferro L, Peano E, Pedrazzini F, Fonio P, et al. Endovascular treatment of visceral artery aneurysms and pseudoaneurysms: our experience. *Radiol Med* 2012;**117**(5):815–30.
- 6 Etezadi V, Gandhi RT, Benenati JF, Rochon P, Gordon M, Benenati MJ, et al. Endovascular treatment of visceral and renal artery aneurysms. *J Vasc Interv Radiol* 2011;**22**(9):1246–53.
- 7 Fankhauser GT, Stone WM, Naidu SG, Oderich GS, Ricotta JJ, Bjarnason H, et al. The minimally invasive management of visceral artery aneurysms and pseudoaneurysms. *J Vasc Surg* 2011;**53**(4):966–70.
- 8 Ferrero E, Ferri M, Viazzo A, Robaldo A, Carbonatto P, Pecchio A, et al. Visceral artery aneurysms, an experience on 32 cases in a single center: treatment from surgery to multilayer stent. *Ann Vasc Surg* 2011;**25**(7):923–35.
- 9 Gabelmann A, Gorich J, Merkle EM. Endovascular treatment of visceral artery aneurysms. *J Endovasc Ther* 2002;**9**(1):38–47.
- 10 Grottemeyer D, Duran M, Park EJ, Hoffmann N, Blondin D, Iskandar F, et al. Visceral artery aneurysms—follow-up of 23 patients with 31 aneurysms after surgical or interventional therapy. *Langenbecks Arch Surg* 2009;**394**(6):1093–100.
- 11 Huang YK, Hsieh HC, Tsai FC, Chang SH, Lu MS, Ko PJ. Visceral artery aneurysm: risk factor analysis and therapeutic opinion. *Eur J Vasc Endovasc Surg* 2007;**33**(3):293–301.
- 12 Lagana D, Carrafiello G, Mangini M, Dionigi G, Caronno R, Castelli P, et al. Multimodal approach to endovascular treatment of visceral artery aneurysms and pseudoaneurysms. *Eur J Radiol* 2006;**59**(1):104–11.
- 13 Marone EM, Mascia D, Kahlberg A, Brioschi C, Tshomba Y, Chiesa R. Is open repair still the gold standard in visceral artery aneurysm management? *Ann Vasc Surg* 2011;**25**(7):936–46.
- 14 Saltzberg SS, Maldonado TS, Lamparello PJ, Cayne NS, Nalbandian MM, Rosen RJ, et al. Is endovascular therapy the preferred treatment for all visceral artery aneurysms? *Ann Vasc Surg* 2005;**19**(4):507–15.
- 15 Tulsyan N, Kashyap VS, Greenberg RK, Sarac TP, Clair DG, Pierce G, et al. The endovascular management of visceral artery

- aneurysms and pseudoaneurysms. *J Vasc Surg* 2007;**45**(2): 276–83.
- 16 Puri S, Nicholson AA, Breen DJ. Percutaneous thrombin injection for the treatment of a post-pancreatitis pseudoaneurysm. *Eur Radiol* 2003;**13**(Suppl 4):L79–82.
  - 17 Roberts KJ, Jones RG, Forde C, Marudanayagam R. Endoscopic ultrasound-guided treatment of visceral artery pseudoaneurysm. *HPB Oxf* 2012;**14**(7):489–90.
  - 18 Sessa C, Tinelli G, Porcu P, Aubert A, Thony F, Magne JL. Treatment of visceral artery aneurysms: description of a retrospective series of 42 aneurysms in 34 patients. *Ann Vasc Surg* 2004;**18**(6):695–703.
  - 19 Roberts K, Revell M, Youssef H, Bradbury AW, Adam DJ. Hypotensive resuscitation in patients with ruptured abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2006;**31**(4):339–44.
  - 20 Ikeda O, Tamura Y, Nakasone Y, Iryou Y, Yamashita Y. Nonoperative management of unruptured visceral artery aneurysms: treatment by transcatheter coil embolization. *J Vasc Surg* 2008;**47**(6):1212–9.
  - 21 Kasirajan K, Greenberg RK, Clair D, Ouriel K. Endovascular management of visceral artery aneurysm. *J Endovasc Ther* 2001;**8**(2):150–5.
  - 22 Rossi M, Rebonato A, Greco L, Citone M, David V. Endovascular exclusion of visceral artery aneurysms with stent-grafts: technique and long-term follow-up. *Cardiovasc Intervent Radiol* 2008;**31**(1):36–42.
  - 23 Chiesa R, Astore D, Guzzo G, Frigerio S, Tshomba Y, Castellano R, et al. Visceral artery aneurysms. *Ann Vasc Surg* 2005;**19**(1):42–8.
  - 24 Shanley CJ, Shah NL, Messina LM. Common splanchnic artery aneurysms: splenic, hepatic, and celiac. *Ann Vasc Surg* 1996;**10**(3):315–22.
  - 25 Guillon R, Garcier JM, Abergel A, Mofid R, Garcia V, Chahid T, et al. Management of splenic artery aneurysms and false aneurysms with endovascular treatment in 12 patients. *Cardiovasc Intervent Radiol* 2003;**26**(3):256–60.
  - 26 Tessier DJ, Abbas MA, Fowl RJ, Stone WM, Bower TC, McKusick MA, et al. Management of rare mesenteric arterial branch aneurysms. *Ann Vasc Surg* 2002;**16**(5):586–90.